SOME CONGENITAL MALFORMATIONS

OF

THE INTESTINAL CANAL.

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Congenital malformations of the intestinal canal are objects of considerable interest to the pathological anatomist. From their study much information may be derived not only of the changes in form, position, and relations which the canal goes through in the course of its development, but of the effects which may be produced on its growth and arrangement by intra-uterine inflammation, affecting either its serous coat or that of the adjacent abdominal wall or viscera.

Three cases of intestinal malformation having recently come under my notice, I purpose relating them to the Society this evening, with some remarks which have been suggested by a consideration of their peculiarities.

The first case to which I shall direct attention is a congenital malformation of the jejunum. The specimen was sent to me about a month ago by Dr Keiller, with a request that I would examine and note its anatomical characters, and communicate them to the Society. It consisted of the abdominal portion of the alimentary canal of a male infant which died twelve days after birth. The stomach was of small size, but normal in shape. Duodenum of usual form, but somewhat dilated. Jejunum, for a distance of 25 inches from its commencement, distended even above the size of the adult intestine; it then appeared to terminate abruptly in a rounded cul-de-sac. The distention was especially well marked for a few inches above the cul-de-sac. A closer examination of the intestine showed, however, that it did not terminate in this abrupt manner, for from the rounded end of this portion of greatly distended jejunum a delicate thread-like structure connected with

the coats of the jejunum, but not communicating with its canal, was prolonged downwards for about 1½ inch. It evidently represented a part of the jejunum atrophied almost to complete disappearance. Below this, again, for 1½ inch, the gut was pervious, and formed a small loop, which had a diameter equal to the stem of a common tobacco-pipe. Beyond this loop was a second constriction, 2 inches long, similar to the one already described. Then the gut, again pervious, formed a second small loop; below which was a third constriction, 1½ inch long, similar to the former ones. The rest of the jejunum and the whole of the ileum were arranged in the usual convoluted manner; their canal was pervious, but their diameter did not exceed that of the two small loops of gut between the three constricted thread-like portions. The mesentery, which corresponded to the three places where the gut presented the greatest amount of constriction, was puckered and shortened, and those portions of it which supported the two short intermediate loops were more or less twisted.[1] I have looked carefully, but

without success, for any appearance of bands of adhesions at these spots, and Dr Thorold, by whom the post-mortem examination was made, has informed me that he saw no traces of old or recent lymph in these localities. An injection which I forced into the trunk of the superior mesenteric artery ran freely into those branches which supplied the bowel above and below the constrictions, but it did not enter into any vessels situated in the three constricted portions and the two small intermediate loops of the jejunum.

[1] *Explanation of Figure.*—*a*, Cul-de-sac. *b, b, b*, Thread-like constricted parts of jejunum. *c, c*, Two short intermediate loops. *d, d*, Shortened mesentery.

The large intestines were normal in their arrangement. Their diameter was about twice that of the ileum and lower end of the jejunum. The ilio-cœcal valve was well formed, and no obstruction existed at its orifice. The biliary and pancreatic ducts opened in the usual way into the duodenum. The jejunum, as far as the cul-de-sac, was distended with a dark brown liquid, which was prevented from passing any lower down, through the constriction already described. The coils of the small intestine contained a bluish-green slimy material, probably inspissated mucus. The valvulæ conniventes were small, the villi well developed. The large intestine contained a material not unlike that found in the lower end of the small. I may also mention that Dr Thorold noticed during the post-mortem examination that the spleen was subdivided into several distinct gland masses, situated partly at the cardiac end, and partly along the great curvature of the stomach.

The chief interest of this case will, I think, rest in the determination of the cause which produced the excessive atrophy I have described, for its position so high up the canal puts it beyond the reach of successful operative interference. To what circumstances, then, are we to ascribe this malformation.

1*st*, Was it produced by a simple diminution of formative power at this spot, during the evolution of the coils of the small intestine out of the single loop of which the canal is at first formed? or,

2*d*, Was it due to a morbid process, probably inflammatory, set up at an early period of embryonic life, which produced a local or limited atrophy of the gut?

That the first named of these supposed causes may occasion atrophy of an organ, or part, is borne out by some of the cases which the records of pathology furnish us with. And such deficiencies in the development of a part especially occur in those localities in which a structure subservient only to the purposes of the fœtal economy shrivels up and altogether or in part disappears. I may, in illustration of this statement, more especially refer to the numerous cases which have now been recorded,[1] in which a contraction of the aorta, amounting in some instances to a complete closure, has been met with at or just below the termination of the arch; localities which embryological research has taught us correspond in the one case to the place of junction of the fifth and fourth vascular arches, in the other to that of the right and left aortic roots. Now, in the natural development of the vessels, both the right aortic root and that part of the fifth left arch which constitutes the ductus arteriosus atrophy and disappear either altogether or partially. And in the cases of contraction of the aorta here referred to, the diminution of the calibre of this vessel seems to have been due to an extension to it of the same process

[1] See an article by Dr Peacock in the Medico Chirurgical Review, 1860 vol. xxv. p. 467; also my Memoir on the Irregularities of the large Bloodvessels, in the same Journal, October 1862, p. 466.

by which the shrivelling up both of the ductus arteriosus and right aortic root is occasioned.

Connected with the canal of the intestine are certain appendicular embryonic structures which, in the ordinary course of development of the entire body, disappear. The vitelline duct is one of these structures. It is continuous with that part of the intestinal canal which subsequently becomes the ileum, its connexion with this portion of the bowel being indicated, even in some adults, by the occasional presence of a diverticulum. More commonly, however, it shrivels up and disappears. Now, it is quite possible to conceive that the atrophy may not be confined to the vitelline duct, but may be extended to the intestine at the spot to which the duct is connected. And thus the canal may become closed, or its further growth beyond the stage which it possessed when the atrophy of the duct commenced either retarded or altogether prevented.

Cases of intestinal obstruction have been recorded which appear to bear out this supposition; cases in which the obstruction had arisen from diminution in the size of the gut, without any evidence of inflammatory mischief. Such, in particular, seems to be the explanation one might give of the origin of an obstruction in the first of two cases recorded by Dr Carver.[1] An infant which died on the fourth day after birth had contraction of the ileum, beginning about 12 inches from its cœcal end, *i. e.*, at the place where with reason one might suppose the vitelline duct had been attached. And it is particularly noted in this case that there were no signs of inflammation either of the peritoneum or bowels. In a second case, an infant which died on the sixth day, there was a similar condition of the ileum, but it was complicated with acute peritonitis, ulceration, and perforation of the lower part of the ileum and cœcum. It appears probable, however, that in this case the atrophy of the intestine preceded the peritonitis rather than that the atrophy was a result of the inflammation.

But though disposed to look upon some cases of intestinal obstruction as occasioned by an extension to the bowel of the process of atrophy, which produces a shrivelling up of some of its embryonic appendicular structures, yet I do not think that the case now before us can be brought into the same category, for the jejunum has not at any period of its development a structure like the vitelline duct connected with its coats or continuous with its canal, serving merely an embryonic purpose and subsequently disappearing. I am inclined, therefore, to regard the inflammatory theory as the one to which the contraction and atrophy of the bowel ought in this case more probably to be referred. And that peritonitis is not unfrequently the cause of various malformations in the intestinal and other abdominal viscera has been satisfactorily established by Professor Simpson, in his well-known memoir "On the Inflammatory Origin of some Varieties of Hernia and

[1] British Medical Journal, 11th August 1860.

Malformation in the Fœtus."[1] The shortened and somewhat puckered condition of the mesentery, corresponding to the thread-like contracted parts of the jejunum, exhibited, I think, indications of the effects of previous inflammation. And although it was noted that there was an absence of bands, either of old or recent adhesions, yet this of itself cannot be accepted as sufficient evidence against the theory of the inflammatory origin of this malformation; for, as was pointed out by Dr Simpson in the memoir already referred to; numerous experiments and observations have shown that old effused lymph or false membrane is often more or less entirely absorbed; and this opinion has since been confirmed by the observations of other pathologists.[2]

But conjoined with this puckered condition of the mesentery, and perhaps in some measure as a consequence of it, the two small loops of jejunum situated between the constricted thread-like portions were twisted upon the axes of their mesenteric attachments. Through this twisting not only was an injurious amount of pressure exercised upon the gut itself, between and immediately above and below these two loops, but the branches of the mesenteric artery passing to these parts of the bowel were evidently so compressed as to prevent the size running along them when I injected the mesenteric trunk.

Through the pressure exercised directly upon these parts of the intestine itself, as well as from the obstructed state of the arteries going to it, we may then in part account, I think, for the extreme degree of atrophy, amounting almost to complete disappearance, which it here presented. Torsion of the small intestine is in itself a matter of some interest; for, from a careful statistical inquiry into the different forms of intestinal obstruction, Dr Brinton has shown[3] that the large, much more frequently than the small, intestine is the seat of such axial twistings.

The difference between the appearance of the intestinal contents in the parts above and below the seat of obstruction was, of course, due to the biliary and pancreatic secretion being cut off from the lower end of the canal. Thus only the part above the obstruction had a bile-tinged colour, the bluish-green contents of the canal below being probably nothing more than inspissated mucus. So that by the division of the intestine into an upper and lower portion, a natural separation, to some extent at least, of the secretion poured into its cavity was produced.

The length of time which the child lived after birth, viz., twelve days, is accounted for by the stricture being so far down the jejunum as to allow of some amount of absorption through the intestinal villi to go on, and this notwithstanding the frequent vomiting which took place after the administration of food.

[1] Edinburgh Medical and Surgical Journal, vol. lii., 1839.
[2] Kirkes, Medical Gazette, April 1849. Paget's Lectures, 2d Edit., p. 282.
[3] Lancet, May 21, 1859.

The cases to which I shall next direct the attention of the Society illustrate two forms of malposition of the cœcum.

In one, an adult male, the cœcum was not placed in its proper fossa, the right iliac, but was displaced upwards, and occupied the adjacent parts of the right lumbar and hypochondriac regions. Here it was not tied down closely to the abdominal wall, but possessed an extensive mesentery. It was therefore movable, and could be thrown across to the left side of the middle line. From it the colon proceeded, and, after making a sharp bend, passed almost immediately into the transverse colon. The ascending colon could hardly be said to exist. Occupying the right iliac fossa, passing upwards through it and the right lumbar region, was the terminal part of the ileum. It possessed in these regions the same relation to the abdominal wall which the cœcum and ascending colon normally have, and was fastened down by the peritoneum just as they are. In the upper part of the right lumbar region the ileum left the wall, acquired a distinct mesentery, passed forwards, and joined the cœcum.

This form of displacement of the cœcum upwards, conjoined with absence of an ascending colon, although rare, has yet occasionally been observed by pathological anatomists. Dr Simpson (p. 35) refers to some cases which have been related in the older records of medicine, and he describes others of a similar nature which came under his own observation. Dr John Reid also saw a case in which the cœcum was placed in the right lumbar region.[1]

This malposition of the commencement of the large intestine is completely to be explained on embryological grounds. It represents a condition which always exists at a certain period of embryonic life.

In the rotation of the two limbs of the primitive loop which represents, in the early weeks of intra-uterine life, the intestinal canal, the posterior limb out of which the cœcum is developed is thrown upwards, so that the cœcum lies in the middle line of the abdomen, or even a little to the left of that line, and in close relation to the under surface of the liver. As the development advances, the cœcum gradually passes into the right hypochondrium, and then descends through the right lumbar region into the right iliac fossa. This descent of the cœcum does not take place until a late period of fœtal life. I have brought with me a fœtus apparently between the fifth and sixth months, in which the cœcum situated in the right lumbar region is in close relation to the under surface of the liver. But its descent may be delayed even to almost the last month of intra-uterine life. In a fœtus at the eighth month, the abdominal cavity of which I opened a short time ago, the cœcum was situated altogether in the right lumbar region, close to the lower end of the right kidney. The right iliac fossa was occupied by a coil of the ileum, which was tied to the back of the fossa by the peritoneum. The testicles had descended into the scrotum.

[1] Edinburgh Medical and Surgical Journal, vol. xlvi. p. 72, 1836.

The ascending colon is therefore the part of the large intestine which is last formed, the space in which it is situated being provided for by the descent of the cœcum into the right iliac fossa.

The other case of malposition of the cœcum consisted in a displacement downwards into the cavity of the pelvis. It occurred in an aged female. The cœcum rested on the floor of the pelvis, and from it the colon ascended through the right iliac and lumbar regions, where it was tied down by the peritoneum, to the right hypochondrium, in which it became continuous with the transverse colon. Owing to the position of the cœcum, the lower end of the ileum also entered the pelvis, and passed to the right side of that cavity to join the large intestine. The cœcum and as much of the ascending colon (about two inches) as was placed in the pelvis were completely surrounded by peritoneum; so that they possessed considerable mobility, and could be thrown over to the left of the pelvis, or even drawn upwards into the cavity of the abdomen proper.

In this case we have an exactly opposite condition to that met with in the one previously described.

Instead of being arrested, the development was excessive, and the cœcum and ascending colon passed through their proper regions to one beyond. It is, I think, a rarer form than the displacement upwards. Dr John Reid has indeed (p. 72) related a closely corresponding case, and he refers to another recorded by Mr Annesley. The malposition would appear to have been occasioned by an excessive growth in the downward direction of the ascending colon, so that the cœcum was projected below its proper region into the pelvis. There was no appearance of peritoneal inflammation, no thickening of the membrane, or adhesions between it and the adjacent parts.

The cases of upward and downward malposition which I have now described are to be distinguished from those one sometimes meets with in which the cœcum is not firmly tied down to the right iliac fossa, but possesses a movable mesentery: for though, in the latter, the cœcum has some amount of mobility, so that it may temporarily undergo a change of place, yet its fossa is not, as in the former cases, permanently occupied by a part of another viscus.

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